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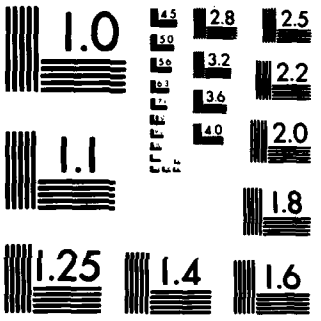

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REPORT NUMBER 2

SLEEP DEPRIVATION AND EXERCISE TOLERANCE

ANNUAL SUMMARY REPORT

BRUCE J. MARTIN, PH.D.

JANUARY 1983

SUPPORTED BY:

U. S. ARMY MEDICAL RESEARCH AND DEVELOPMENT COMMAND
FORT DETRICK, FREDERICK, MARYLAND 21701

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INDIANA UNIVERSITY SCHOOL OF MEDICINE
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30 hours of sleep loss failed to alter physiological responses to prolonged heavy exercise. At the same time it decreased significantly the ability to tolerate such exercise. These results suggest that psychological effects of sleeplessness may ultimately be most significant for subjects in exercise.

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FOREWORD

For the protection of human subjects the investigator(s) have adhered to policies of applicable Federal law 45 CFR 46.

BODY OF REPORT

Statement of the Problem:

Sleep deprivation occurs commonly in both civilian and military contexts. Despite this, little is known of the effects of sleep loss on physiological function. Such knowledge could become particularly useful when severe exercise occurs after sleep deprivation.

Background:

The effects of sleep loss on exercise have been only slightly examined. In some instances, sleep loss has been combined with exercise stress and caloric restriction, so that any subsequent effects seen in exercise are difficult to ascribe to a single stressor (1, 2). Other more direct studies have used inexact (3) measures of exercise responses. Still other studies of sleep loss have focused upon resting physiological responses (4) or psychological variations (5).

Our previous work, reported in an earlier Annual Report, examined physiological responses to exercise after one night (30 hours) of sleep deprivation. These studies showed that while submaximal and maximal oxygen uptake were identical with or without prior sleep, tolerance of prolonged heavy exercise was reduced (6, 7). It also appeared that subjective assessment of work intensity was unchanged by a 30-hour sleep loss period (8).

Approach to the Problem:

These previous investigations suggest that sleep loss reduces prolonged exercise tolerance for reasons that remain unclear. Equally unspecified was the amount of sleep loss necessary for any performance deficit. We thus decided to extend the sleep loss period, and to investigate a wider range of physiological responses to exercise, in an attempt to better define the physiological and exercise performance effects of sleep deprivation.

Results and Discussion:

In this year of the Contract, one study has been completed that will be described in detail. In this study, 8 subjects walked until exhausted at a work rate requiring about 80% of their maximal oxygen uptake. On one occasion, they walked after a 50 hour sleepless period, while on the other they exercised after control sleeping conditions.

On each occasion, we measured oxygen uptake, CO_2 production rates, minute ventilation, and heart rate each 15 minutes during exercise, and blood lactate levels, and levels of norepinephrine, epinephrine, and dopamine in peripheral venous blood once each after 15 min of exercise. Also, we monitored core (rectal) and inner thigh skin temperatures throughout each walk.

We found that the 50-hour sleepless period significantly reduced work time to exhaustion by about 20% ($P<0.01$). This decline was very nearly significantly greater than that seen in our previous study utilizing a 36 hour sleepless period ($0.05 < P < 0.10$). This result suggests that sleep loss may act in drug-like fashion, with its effects on exercise tolerance appearing in dose-dependent fashion. In these, as in previous studies, subjects were not told their work times nor were they allowed to watch a clock during exercise.

Despite these marked decreases in work tolerance, no physiological changes induced by sleep loss were measurable in exercise. Oxygen uptake, minute ventilation, and heart rate were identical throughout work in the two situations. Similarly, core and inner thigh skin temperatures were virtually the same with or without sleep loss. Also, blood lactate levels, and the concentration of norepinephrine, epinephrine, and dopamine were similar in both conditions. These results suggest that sleep loss reduces work tolerance in prolonged exercise without producing a measurable physiological effect. These data thus suggest that sleeplessness may well act through psychological effects to alter prolonged exercise tolerance.

Conclusions:

Based on two years of work, we conclude that sleep deprivation acts in drug-like fashion to reduce the ability to tolerate prolonged heavy exercise: greater sleep loss results in a proportionally greater decline in work performance. However, we have as yet failed to identify a physiological mechanism for this decline, and thus at present feel that psychological, not physiological, factors underlie this effect.

Recommendations:

Sleep loss of even 50 hours duration remains only a mildly potent inhibitor of exercise performance: its effects are quantitatively similar to those induced by prior exercise or significant dietary alteration. However, we have investigated required exercise: were exercise to be initiated by the subject in a self-motivated manner, it might well be that greater deleterious effects on exercise would be uncovered.

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Publications:

Martin, B. J., and H.-I. Chen. Sympathoadrenal responses to exercise after sleep loss. Med. Sci. Sports Exercise (submitted).

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